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Review Article

Sinusoidal Obstruction Syndrome/Veno-Occlusive Disease in Adult Patients after Hematopoietic Stem Cell Transplantation: A Review of Diagnostic Accuracy of Liver Stiffness Measurement by Elastography

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ABSTRACT

The recent European Society for Blood and Marrow Transplantation (EBMT) criteria for diagnosis of Sinusoidal Obstruction Syndrome/Veno-Occlusive Disease (SOS/VOD) provided a new classification of "probable" SOS/VOD to improve early diagnosis and ensure prompt therapy with defibrotide. Transient Elastography (TE) and Shear Wave Elastography (SWE) have emerged for early and accurate diagnosis of SOS/VOD after Hematopoietic Stem Cell Transplantation (HSCT). In this review, the author outlined the current knowledge and trends of SOS/VOD in adult patients after HSCT for diagnostic accuracy of Liver Stiffness Measurement (LSM) by elastography. The MEDLINE/PubMed and EMBASE data-bases were systematically accessed for studies using elastography in adult patients after HSCT. In addition, the new biomarker studies for endothelial damage such as VODcheck and Circulating Endothelial Cell (CEC) have been also described. Based on the evidence, the increased LSM in the acute phase of SOS/VOD probably may be attributable to the changes caused by inflammation rather than fibrosis. SWE and TE may be promising tools for early accurate diagnosis, disease severity, and follow-up for SOS/VOD. The estimation of endothelial damage using biomarker examination might be a potential procedure for early detection and treatment of SOS/VOD after HSCT.

Keywords: Sinusoidal obstruction syndrome/veno-occlusive disease; Hematopoietic stem cell transplantation; Liver stiffness measurement; Elastography; Endothelial biomarker

INTRODUCTION

The recent EBMT criteria for diagnosis of SOS/VOD provided a new classification of "probable" SOS/VOD to improve early diagnosis and make certain of prompt therapy with defibrotide [1]. In addition to the Ultrasound (US), the utility of TE and SWE has emerged for early and accurate diagnosis of SOS/VOD after HSCT [2,3]. In this review, the current knowledge and trends of SOS/VOD in adult patients after HSCT for diagnostic accuracy of LSM by elastography have been summarized. Additionally, the recent studies have reported endothelial biomarker test for the evaluation of SOS/VOD after HSCT including VODCheck and CEC study [4,5]. The novel biomarkers of endothelial damage for SOS/VOD following HSCT have been also described.

LITERATURE REVIEW

Mechanism of SOS/VOD development by cytotoxic drugs

The first step of pathogenesis in SOS/VOD is the damage of sinusoidal endothelial cell of the liver leading to loss of endothelial cell cohesions. In result, the detachment of the endothelial cells and subsequent post-sinusoidal obstruction have developed [6]. Conditioning regimens showed a significant role in the pathogenesis of development of SOS/VOD due to higher plasma levels of cytotoxic drugs including Busulfan (BU) or Cyclophosphamide (CY). It is known that chemotherapy agents are metabolized by the cytochrome P450 complex, inducing the toxic metabolites. In result, they were converted to non-toxic types by the Glutathione (GSH) enzymatic system. Additionally, a GSH S-transferase M1 null genotype decreasing the detoxifying capacity of the liver parenchyma predisposes to SOS. Endothelial cells following HSCT exhibit characteristic features of procoagulant and proinflammatory condition confirming by the presence of increased levels of circulating markers of endothelial activation. Endothelial cell detachment seems to be associated with Nitric Oxide (NO) deficiency by after-conditioning toxicity leading to increased endothelial cell production of Matrix Metalloproteinase

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9 (MMP-9). The role of MMP-9 in the pathogenesis of SOS/VOD has been suggested [6].

Updated criteria of EBMT for SOS/VOD after HSCT

In 2016, Seattle criteria established in 1984 and Baltimore criteria in 1987 have been replaced by the revised criteria for the diagnosis of SOS/VOD proposed by the European Society for Blood and Marrow Transplantation (EBMT) describing the revised diagnosis and severity criteria for SOS/VOD patients [7]. It is known that there are three risk factors for SOS/VOD including transplant-related, patient and disease-related, and hepatic-related factors [7]. Regarding transplant-related risk factors, the risk for SOS/VOD development depends on the conditioning regimen intensity and the drugs. Compared with the Reduced Intensity Conditioning (RIC), it is higher incidence following high-dose Busulfan (BU) or Total Body Irradiation (TBI)-based conventional Myeloablative Conditioning (MAC). High-dose TBI, and/or a combination of BU and CY also lead to an increased occurrence of SOS/VOD [7]. Although SOS/VOD with mild to moderate state resolves within a few weeks, the most severe type tends to result in Multiorgan Dysfunction (MOD). Previously, classical SOS/VOD was diagnosed by elevated bilirubin level, and two of the following criteria such as painful hepatomegaly, weight gain >5%, and ascites. Beyond day 21 after HSCT, it was diagnosed as classical SOS/VOD or histologically proven or by the presence of two or more of the following criteria including clinical and biochemical findings and hemodynamical and/or US evidence of SOS/VOD [7,8]. An international consensus guideline for the treatment prevention of SOS/VOD as a position statement has been also proposed focusing on prophylactic, preemptive, and curative treatment for SOS/VOD in adult patients [9]. Furthermore, Mohty et al. updated the previous classification and refined as probable, clinical, and proven SOS/VOD at diagnosis in adult patients [1]. They updated the criteria with the addition of a new category such as probable SOS/VOD showing that two of more of the following 5 criteria, hyperbilirubinemia, painful hepatomegaly, weight gain, ascites, ultrasound and/or elastography suggestive of SOS/VOD [1]. They stated an accurate definition of MOD for SOS/VOD severity grading including mild, moderate, severe, and very severe based on Sequential Organ Failure Assessment (SOFA) score. Gray scale and color Doppler US showed unspecific hepatosplenomegaly, reversed portal vein flow or high Resistance Index (RI) of the hepatic artery, while Contrast-Enhanced Ultrasound (CEUS) or LSM, a few studies have exhibited promising results for artery diagnosing SOS/VOD [10]. Ultrasound techniques including combination of Doppler ultrasound with elastography and CEUS methods should be assessed for accurate diagnosis and follow-up of SOS/VOD [8]. It is known that elastography is relevant for inclusion in the SOS/VOD diagnosis criteria due to sensitivity and specificity for SOS. While, LSM is a useful procedure for assessment such as fibrosis, acute inflammation, congestion, and cholestasis. It is an also validated tool for evaluation for Hepatic Venous Pressure Gradient (HVPG) and portal hypertension [11].

Assessment of LSM in SOS/VOD before and after HSCT

A chemotherapy dependent toxic impairment to hepatic sinusoidal endothelial cells leads to a local inflammation and migration of blood cells into the space of Disse exhibiting an obstruction of the sinusoidal microcirculation. The previous reports described the mechanism and LSM as a novel predictor by elastography in patients with oxaliplatin-induced SOS [12,13]. It is known that patients with splenomegaly after receiving oxaliplatin had significantly higher mean elasticity measurements by fibroscan at 3 and 6 months respectively suggesting that LSM could be noninvasively utilized for predicting oxaliplatin-induced hepatotoxicity [14]. LSM is a technique for diagnosing liver fibrosis in chronic liver diseases such as viral hepatitis or NAFLD, Metabolic Dysfunction-Associated Fatty Liver Disease (MAFLD), and Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) [8,15]. The diagnosis of SOS/VOD was based on the revised EBMT 2023 criteria including Doppler ultrasound and elastography for early detection of venous stasis, as well as stratification of cases according to grades of diagnostic certainty including probable, clinical, and proven category [1]. Regarding color Doppler US, a reversal or reduction in portal venous flow is useful in diagnosing hepatic congestion with SOS/VOD. With regards to LSM methods, TE, point shear wave with Acoustic Radiation Force Impulse (ARFI), and two-dimensional real-time Shear Wave Elastography (2D-SWE) have been exhibited [16]. LSM is calculated by SWE of the elastic properties of liver tissue. Previous study showed that SOS status has been accompanied by an increase in shear wave velocity suggesting that this procedure may contribute to diagnosing the development of SOS following HSCT [8]. Previous study showed the ultrasound elastography measurement by 2D-SWE before transplantation, at day +7 and day +14 demonstrating that 2D-SWE at day +14 exhibited an early detection for SOS with a statistical significance and improved sensitivity, specificity and positive predictive value over the Seattle, Baltimore or EBMT scores [17]. A significant increase in 2D-SWE by Debureaux et al. was observed in patients with SOS suggesting that a 2D-SWE measurement above 8.1 kPa with improved sensitivity and specificity of 75 and 99% respectively day +14 after allo-HSCT represents non-invasively potential and reproducible method for early and accurate diagnosis for SOS [17]. Davidov et al. described that none of the patients with the high-risk group with a LSM less than 7 kPa by TE developed SOS after HSCT [16]. They found that there was a positive correlation between LSM and the diagnosis of SOS with statistical significance ($r=0.66$, $p=0.007$) using Spearman rank correlation coefficient. They indicated that a LSM cut-off of 7.5 kPa had a sensitivity and specificity of 80 and 86% respectively for diagnosing SOS suggesting that before HSCT, LSM can help determine the conditioning treatment in patients with high-risk for SOS and after HSCT it can make help in early diagnosing for SOS [16]. Some studies have also described the role of LSM for diagnosis of SOS in adult patients following HSCT [8,18-20]. Schulz et al. have reported three patients with an increase in shear wave velocity describing that SWE method may contribute to diagnosing SOS/VOD post-HSCT [8]. Previous study using TE showed that LS exceeded 17.4 kPa with sensitivity and specificity of 100 and 90%, respectively indicating that LSM may contribute to diagnosis for SOS/VOD post-HSCT [19]. In addition, a case of the late-onset SOS diagnosed pathologically has been reported exhibiting that the histological features were consistent with SOS/VOD [21]. While, the conditioning regimens serve as a potential role in the pathogenesis of SOS development suggesting that higher levels of cytotoxic drugs such as metabolities of BU or CY are associated with the increased risk of SOS [22]. To diagnose the accuracy of LSM for SOS/VOD after HSCT, multicentre diagnostic clinical trial across 25 centers including both adult and pediatric patients has been investigated describing that a diagnostic algorithm was proposed, showing >95% sensitivity and specificity, with a 6 kPa roue-out and 25 kPa rule-in cut-off value [2]. LSM

can also distinguished SOS from other complications within +100 days after HSCT, demonstrating that LSM is a significant potential diagnostic tool, contribution of differential diagnosis, and therapy response for SOS. Whereas transient and shear wave elastography for the detection of SOS in patients receiving HSCT from a systematic review and meta-analysis including ten studies, 100 patients diagnosed with SOS have been reported, suggesting that elastography exhibited non-invasively promising tool for early detection with significant increase in Shear Wave Velocity (SWV) and liver stiffness values [3].

The US is an operator-dependent method and some criteria are not recommended as diagnostic criteria in children as previously described [3]. It is known that elastography is a quantitative, reproducible, and available diagnostic tool showing that its characteristics make it suitable in pediatric patients treated with HSCT [3]. Several reports of LSM values using elastography have been also reported in the pediatric study [23-27]. A new classification of the diagnosis and severity by EBMT and imaging findings including elastography for SOS/VOD in pediatric patients has been also reported [28,29]. SWE can measure the US waves propagation velocity in meters per second (m/s) showing a well-established procedure to detect the grade of the liver fibrosis in chronic liver disease such as NAFLD [3,27]. Whereas the TE value represented Young's modules in kilo pascals and it has relatively high failure rates and small study populations [3,27]. According to the previous study, LSM by SWE may be increased not only due to fibrosis but to various conditions such as liver inflammation, congestion, portal hypertension, and cholestasis [30]. With regards to the histological changes in SOS/VOD, fibrosis is not found at the acute phase of toxic damage, whereas dense perivascular fibrosis persists over weeks to months [31]. Previous study described that the subsequent decrease value in LSM following therapy might be attributable to changes by inflammation, congestion, or portal hypertension rather than fibrosis [27]. In this review, the current knowledge of SOS/VOD after HSCT for the assessment of LSM in adult patients has been outlined (Tables 1 and 2). Based on the evidence, the increased LSM in the acute phase of SOS/VOD and the decreased value after therapy have shown, suggesting that this phenomenon probably may be attributed to the alterations caused by inflammation rather than fibrosis. The results provided that SWE and TE may be potential tools for early diagnosis and disease severity indicator of SOS/VOD.

Table 1: Liver stiffness measurement for sinusoidal obstruction syndrome/veno-occlusive disease in adult patients after HSCT.

Study	Design	HSCT patients (N)	SOS patients (N)
Colecchia et al. [20]	Prospective	78	4
Debureaux et al. [17]	Prospective	146	6
Schulz et al. [8]	Prospective	63	3
Ozkan et al. [18]	Retrospective	31	2
Davidov et al. [16]	Prospective	17	10
Inoue et al. [19]	Retrospective	86	14

Table 2: Liver stiffness measurement for sinusoidal obstruction syndrome/veno-occlusive disease in adult patients after HSCT.

Study	LSM procedure	LSM at diagnosis kPa	AUC	Sensitivity	Specificity
Colecchia et al. [20]	TE	14.1-34.3	0.997	75%	98.7%
Debureaux et al. [17]	2D-SWE	8.1 (cut-off value)	0.84	75%	99%
Schulz et al. [8]	2D-SWE	58.8, 8.9, 13.9	NA	NA	NA
Ozkan et al. [18]	TE	55, 17.7	0.569	100%	55.17%
Davidov et al. [16]	TE	7.5 (cut-off value)	0.89	80%	86%
Inoue et al. [19]	TE	>17.4	0.9663	100%	90.3%

NA: Not Applicable

Additionally, the previous study provided that several pathway analyses demonstrated major gene upregulation in six pathways in oxaliplatin-induced SOS [32]. The author previously described that gene expression revealed biological pathways including oxidative stress, inflammation, hepatic fibrosis/HSC activation, coagulation, angiogenic, and hypoxic factors for oxaliplatin-induced SOS. In particular, inflammatory pathway is significantly updated, suggesting a main driving factor of hepatotoxicity by oxaliplatin chemotherapy [33]. Similar to the development of oxaliplatin-induced SOS, LSM value may reflect inflammation in patients with SOS/VOD following HSCT.

Novel biomarker studies of endothelial damage for SOS/VOD after HSCT

Regarding the pathophysiology of SOS/VOD, the injury of the Sinusoidal Endothelial Cells (ECs) and hepatocytes have been demonstrated. The activation of ECs increases expression of adhesion molecules (VCAM-1, E-selectin). A key role is served by the angiopoietin (Ang-1) and angiopoietin (Ang-2). This imbalance between Ang-1 and Ang-2 contributes to increase of vascular permeability, leakage into the space of Disse, and narrowing of the sinusoid reading to fibrosis and vasoconstriction [34]. Larue et al. described the key risk factors, new diagnostic procedures, and therapeutic strategies for SOS/VOD. In addition, they also emphasized the early use of defibrotide as the reference therapy for severe SOS/VOD [34]. Recent studies have reported the endothelial biomarker test for the evaluation of SOS/VOD after HSCT. VODCheck including Hyaluronan (HA), Angiopoietin-2 (ANG-2), and Thrombomodulin (TM), so-called a biomarker-based prognostic test for the assessment of SOS/VOD risk was significantly prognostic of SOS/VOD risk after HCT at day 7, and after adjustment for confounding factors including age, SOS/VOD risk state, primary disease, and ozogamicin therapy, it remained prognostic. VODCheck was also significantly prognostic of SOS/VOD risk on days 1 and 15, suggesting that this test was validated as an independent predictive marker of SOS/VOD risk within 15 days after HCT by a multivariate analysis [4]. Meanwhile, the emerging evidence indicated that cellular-based markers including CECs, Endothelial Progenitor Cells (EPCs), and Extracellular Vesicles (EVs) exhibit a newl tool to evaluating endothelial function [35]. Regarding HSCT, a significant increase in CEC numbers compared to baseline has been related to the administration and type of the conditioning [35]. The CEC level can be a potential biomarker for diagnosing SOS/VOD and detecting patients with late-onset SOS/VOD [5]. CECs were detected using the Food and Drug Administration-approved CellSearch system and were defined as CD146+, CD105+, DAPI+, or CD45-. The result provided that a baseline CEC count >17/mL was associated with an elevated risk of SOS/VOD, along with bilirubin level >1.5 mg/mL and a haploidentical donor HSC source [5]. Results showed that a relative increase in CEC count >1.5-fold was confirmed as a predictive factor with the development of late-onset SOS/VOD by a multivariate analysis, along with a high Endothelial Activation and Stress Index (EASIX) score at engraftment [5]. The estimation of endothelial function using biomarker examination might be a potential procedure for early detection, disease severity, and treatment of SOS/VOD after HSCT. Further investigations for endothelial function are needed to elucidate in the patients with SOS/VOD after HSCT. Meanwhile, endothelial function test included Flow-Mediated Vasodilation (FMD) and Arterial Stiffness (AS) such as Brachial-Ankle Pulse Wave Velocity (ba-PWV). Endothelial dysfunction is the first step for atherosclerosis condition therefore FMD and Nitroglycerin-Mediated Vasodilation (NMD) tests are crucial procedures for evaluation of atherosclerosis [36-38]. The author suggested the assessment of vascular endothelial and smooth muscle cell (SMC) function using FMD and NMD procedures for atherosclerosis status in the setting of oxaliplatin-based chemotherapy [33]. Regarding systemic atherosclerosis status after HSCT, previous study indicated that endothelial function using FMD was decreased in patients with hematologic malignancies about 22 days after HSCT [39]. Evidence has provided that chemotherapy, radiation proteasome inhibitors, and anti-VEGF therapies promote pathologic structural and functional changes of the vascular wall reflecting arterial stiffness [40]. The mechanisms underlying cancer therapy-related vascular toxicities have emerged including the proof of oxidative stress and inflammation by NO signaling, the elastin/collagen ratio, and SMC senescence [40]. A recent meta-analysis has shown increased AS in patients with cancer exhibiting a pathophysiological link between cancer therapy and AS [41]. Previous studies demonstrated the reduction in endothelium-dependent vasodilation of the brachial artery and microcirculatory vessels in patients with cancer receiving HCT [40, 42].

DISCUSSION

Compared with the RIC, it is higher incidence following high-dose BU or TBI-based MAC. High-dose TBI, and/or a combination of BU and CY also lead to an increased occurrence of SOS/VOD. The recent EBMT criteria for diagnosis of SOS/VOD provided a new classification of "probable" SOS/VOD to improve early diagnosis and make certain of prompt therapy with defibrotide [1]. In addition to the US, the utility of TE and shear elastography has emerged for early and accurate diagnosis of SOS/VOD after HSCT. In this review, SOS/VOD in adult patients after HSCT for diagnostic accuracy of LSM by elastography has been currently outlined (Tables 1 and 2). A significant increase in 2D-SWE by Debureaux et al. was observed in patients with SOS suggesting that a 2D-SWE measurement above 8.1 kPa with improved sensitivity and specificity of 75 and 99% respectively at day +14 after allo-HSCT represents non-invasively potential and reproducible method for early and accurate diagnosis for SOS [17]. Davidov et al. have reported that a LSM cut-off of 7.5 kPa had a sensitivity and specificity of 80 and 86% respectively for diagnosing SOS suggesting that before HSCT, LSM can help determine the conditioning treatment in patients with high-risk for SOS and after HSCT it can make help in early diagnosing for SOS [16]. To diagnose the accuracy of LSM for the diagnosis of VOD/SOS after HSCT, multicentre diagnostic clinical trial across 25 centers including both adult and pediatric patients has been investigated describing that a diagnostic algorithm was proposed, showing > 95% sensitivity and specificity, with a 6 kPa roue-out and 25 kPa rule-in cut-off value [2]. LSM can also distinguished SOS from other complications within +100 days after HSCT, demonstrating that LSM is a significant potential diagnostic tool, contribution of differential diagnosis, and therapy response for SOS. Whereas transient and shear wave elastography for the detection of SOS in patients receiving HSCT from a systematic review and meta-analysis including ten studies, 100 patients diagnosed with SOS have been reported, suggesting that elastography exhibited non-invasively promising tool for early detection with significant increase in SWV, and liver stiffness values [3]. Based on the evidence, the increased LSM in the acute phase of SOS/VOD probably may be attributable to the changes caused by inflammation rather than fibrosis. The

results provided that SWE and TE may be potential tools for early diagnosis and disease severity indicator of SOS/VOD. In addition, similar to the development of oxaliplatin-induced SOS, LSM value may reflect inflammation in patients with SOS/VOD following HSCT. Meanwhile, the recent studies have reported endothelial biomarker test for the evaluation of SOS/VOD after HSCT. VODCheck was also significantly prognostic of SOS/VOD risk on days 1, 7, and 15, suggesting that this test was validated as an independent predictive marker of SOS/VOD risk within 15 days after HCT by a multivariate analysis [4]. Regarding CEC study, results showed that a relative increase in CEC count >1.5-fold was confirmed as a predictive factor with the development of late-onset SOS/VOD in a multivariate analysis, along with a high EASIX score [5]. The estimation of endothelial function using biomarker examination might be a potential procedure for early detection, disease severity, and treatment of SOS/VOD after HSCT. Further investigations for endothelial damage using biomarkers are needed to elucidate in patients with SOS/VOD after HSCT.

CONCLUSION

Based on the evidence, the increased liver stiffness measurement in the acute phase of SOS/VOD after HSCT probably may be attributed to the changes caused by inflammation rather than fibrosis. The results provided that shear wave elastography and transient elastography may be promising tools for early accurate diagnosis, disease severity, and follow-up for SOS/VOD post-HSCT. The assessment of endothelial dysfunction using biomarker examinations might be a potential procedure for early detection and treatment of SOS/VOD after HSCT.

CONFLICT OF INTEREST

Author declares that I have no conflicts of interest.

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